AMENDMENTS TO THE CLAIMS:

*This listing of the claims below **will** replace all **prior** versions and listing of claims in this application.*

1. (Previously presented) A method for proliferating cardiomyocytes comprising a step of introducing

(a) cyclin,

(b) cyclin-dependent kinase, and

(c) one or a plurality of a gene encoding a factor that inhibits the production, function or action of Cip/Kip family protein, into cardiomyocytes *in vitro*, and

a step of subsequently culturing or maintaining said cells.

2. (Previously presented) A method for proliferating cardiomyocytes comprising a step of introducing

(a) cyclin,

(b) cyclin-dependent kinase, and

(c) one or a plurality of a gene encoding a factor that inhibits the production, function or action of Cip/Kip family protein, into cardiomyocytes *in vitro*, and

a step of subsequently culturing said cells.

3. (Withdrawn) A method for proliferating cardiomyocytes comprising a step of introducing

(a) cyclin,

(b) cyclin-dependent kinase, and

(c) one or a plurality of a gene encoding a factor that inhibits the production, function or action of Cip/Kip family protein, or a nucleic acid that inhibits the production of Cip/Kip family protein, into cardiomyocytes *in vivo*, and

a step of subsequently maintaining said cells.

4. (Previously presented) The method of claim 1, wherein said cyclin is a cyclin that activates CDK4 or CDK6 of mammals.

5. (Original) The method of claim 4, wherein said cyclin is cyclin D of mammals.

6. (Previously presented) The method of claim 1, wherein said cyclin-dependent kinase is a cyclin-dependent kinase to be activated by cyclin D.

7. (Previously presented) The method of claim 6, wherein said cyclin dependent kinase is CDK4 or CDK6.

8. (Previously presented) The method of claim 1, wherein the Cip/Kip family protein is $p27^{Kip1}$.

9. (Previously presented) The method of claim 1, wherein the factor that inhibits the production, function, or action of Cip/Kip family protein is a factor with an action to promotes the degradation of the Cip/Kip family protein.

10. (Original) The method of claim 9, wherein the factor with an action to promote the degradation of the Cip/Kip family protein is a component of ubiquitin ligase.

11. (Previously presented) The method of claim 10, wherein the component of ubiquitin ligase is an F-box factor that binds to the Cip/Kip family protein.

12. (Original) The method of claim 11, wherein the F-box factor capable of binding to the Cip/Kip family protein is Skp2.

13. (Withdrawn) The method of claim 1, wherein the nucleic acid that inhibits the production of Cip/Kip family protein is siRNA specific to a gene encoding the Cip/Kip family protein.

14. (Withdrawn) The method of claim 13, wherein the nucleic acid that inhibits the production of Cip/Kip family protein is siRNA specific to the $p27^{Kip1}$ gene.

15. (Previously presented) The method of claim 1, comprising introducing the genes into cardiomyocytes, using a viral vector or liposome.

16. (Previously presented) The method of claim 1, wherein at least one of the cyclin gene and cyclin-dependent kinase gene is tagged with a nucleotide sequence encoding a nuclear localization signal.

17. (Previously presented) A vector comprising

(a) a cyclin gene

(b) a cyclin-dependent kinase gene, and

(c) one or a plurality of a gene encoding a factor that inhibits the production, function, or action of Cip/Kip family protein.

18. (Previously presented) The vector of claim 17, wherein the cyclin is a cyclin that activates CDK4 or CDK6 of mammals.

19. (Original) The vector of claim 18, wherein the cyclin is cyclin D of mammals.

20. (Previously presented) The vector of claim 17, wherein the cyclin-dependent kinase is a cyclin-dependent kinase to be activated by cyclin D.

21. (Original) The vector of claim 20, wherein the cyclin-dependent kinase is CDK4 or CDK6.

22. (Previously presented) The vector of claim 17, wherein the factor that inhibits the production, function, or action of Cip/Kip family protein is a factor with an action to promote the degradation of the Cip/Kip family protein.

23. (Original) The vector of claim 22, wherein the factor with an action to promote the degradation of the Cip/Kip family protein is a component of ubiquitin ligase.

24. (Original) The vector of claim 23, wherein the component of ubiquitin ligase is an F-box factor capable of binding to the Cip/Kip family protein.

25. (Original) The vector of claim 24, wherein the F-box factor capable of binding to the Cip/Kip family protein is Skp2.

26. (Withdrawn) The vector of claim 17, wherein the nucleic acid that inhibits the production of Cip/Kip family protein is siRNA specific to a gene encoding the Cip/Kip family protein.

27. (Withdrawn) The vector of claim 26, wherein the nucleic acid that inhibits the production of Cip/Kip family protein is siRNA that is specific to $p27^{Kip1}$ gene.

28. (Withdrawn) The vector of claim 17, wherein at least one of the cyclin gene and cyclin-dependent kinase gene is tagged with a nucleotide sequence encoding a nuclear localization signal.

29. (Withdrawn) A pharmaceutical composition for use in a treatment of cardiac disorder comprising the vector of claim 17.

30. (Withdrawn) The pharmaceutical composition of claim 29, wherein the cardiac disorder is myocardial infarction, ischemic heart disease, congestive heart failure, hypertrophic cardiomyopathy, dilated cardiomyopathy, myocarditis, or chronic heart failure.

31. (Previously presented) Cardiomyocyte obtained by the method of claim 1.

Claims 32-33 (Canceled)

34. (New) A method for proliferating cardiomyocytes that have withdrawn from the cell cycle comprising a step of introducing

(a) cyclin,

(b) cyclin-dependent kinase, and

(c) one or a plurality of a gene encoding a factor that inhibits the production, function or action of Cip/Kip family protein, into cardiomyocytes *in vitro*, wherein said cardiomyoctes have withdrawn from the cell cycle, and

a step of subsequently culturing or maintaining said cells.

35. (New) The method of claim 34, wherein the cardiomyocytes are adult cardiomyocytes.